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Term	Documents
(5 AND 6).PGPB,USPT,USOC,EPAB,JPAB,DWPI,TDBD.	34
(L6 AND L5).PGPB,USPT,USOC,EPAB,JPAB,DWPI,TDBD.	34

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DATE: Friday, May 21, 2004 Printable Copy Create Case

Set Name side by side	Query	Hit Count	Set Name result set
DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI,TDBD; THES=ASSIGNEE; PLUR=YES; OP=AND			
L8	L6 and L5	34	L8
L7	L6 same (vector or transfect or transfection)	24	L7
L6	(CD154 or CD40L) same (skin or muscle or fibroblast or endothelial or neuronal or bone or cartilage or liver or kidney or spleen)	413	L6
L5	L4 and L3	108	L5
L4	(cell or organ or tissue) adj (transplant or transplantation)	15902	L4
L3	L2 and (vector)	187	L3
L2	(CD154) and (CD40 adj ligand)	220	L2
L1	Heath-andrew-W$.in.	8	L1

END OF SEARCH HISTORY

h e b b cg b e e ch

PALM INTRANET

Day : Friday
Date: 5/21/2004

Time: 14:46:24

Inventor Name Search

Enter the **first few letters** of the Inventor's Last Name.
Additionally, enter the **first few letters** of the Inventor's First name.

Last Name	**First Name**	
Heath	Andrew	Search

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h e eb cg b e f e

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### Status: Path 1 of [Dialog Information Services via Modem]

### Status: Initializing TCP/IP using (UseTelnetProto 1 ServiceID pto-dialog)
Trying 31060000009999...Open

DIALOG INFORMATION SERVICES
PLEASE LOGON:
 ******** HHHHHHHH SSSSSSSS?
### Status: Signing onto Dialog
 ********
ENTER PASSWORD:
 ******** HHHHHHHH SSSSSSSS? ********
Welcome to DIALOG
### Status: Connected


Dialog level 04.09.00D

Last logoff:  21may04 12:51:23
Logon file001 21may04 15:13:39
KWIC is set to 50.
HILIGHT set on as '*'
* ALL NEW CURRENT YEAR RANGES HAVE BEEN * * *
* * * INSTALLED   * * *
*
File   1:ERIC 1966-2004/May 18
       (c) format only 2004 The Dialog Corporation

      Set  Items  Description
      ---  -----  -----------
Cost is in DialUnits
?b 155, 5, 73
       21may04 15:13:47 User259876 Session D625.1
            $0.29    0.083 DialUnits File1
     $0.29  Estimated cost File1
     $0.03  TELNET
     $0.32  Estimated cost this search
     $0.32  Estimated total session cost   0.083 DialUnits


SYSTEM:OS  - DIALOG OneSearch
  File 155:MEDLINE(R) 1966-2004/May W3
        (c) format only 2004 The Dialog Corp.
*File 155: Medline has been reloaded. Accession numbers
have changed. Please see HELP NEWS 154 for details.
  File   5:Biosis Previews(R) 1969-2004/May W3
        (c) 2004 BIOSIS
  File  73:EMBASE 1974-2004/May W2
        (c) 2004 Elsevier Science B.V.

      Set  Items  Description
      ---  -----  -----------
?s (CD154 or CD40L) (s) (skin or muscle or fibroblast or endothelial or neuronal or bon
e or cartilage or liver or kidney or spleen)
           1987  CD154
           4648  CD40L
         884721  SKIN
        1286110  MUSCLE
         208554  FIBROBLAST
         319563  ENDOTHELIAL
         346166  NEURONAL
        1063104  BONE
         115621  CARTILAGE
        1491105  LIVER
        1049164  KIDNEY
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          309393  SPLEEN
      S1    1378  (CD154 OR CD40L) (S) (SKIN OR MUSCLE OR FIBROBLAST OR
                  ENDOTHELIAL OR NEURONAL OR BONE OR CARTILAGE OR LIVER OR
                  KIDNEY OR SPLEEN)
?s s1 (s) (transfection or transformation or infection)
            1378  S1
          169422  TRANSFECTION
          317361  TRANSFORMATION
         2459237  INFECTION
      S2     152  S1 (S) (TRANSFECTION OR TRANSFORMATION OR INFECTION)
?s s2 (s) (recombinant)
             152  S2
          542288  RECOMBINANT
      S3      19  S2 (S) (RECOMBINANT)
?rd
...completed examining records
      S4       8  RD (unique items)
?t s4/3,k/all
```

4/3,K/1 (Item 1 from file: 155)

DIALOG(R)File 155:MEDLINE(R)
(c) format only 2004 The Dialog Corp. All rts. reserv.

14253416 PMID: 10072446

Two neutralizing human anti-RSV antibodies: cloning, expression, and characterization.

Heard C; Brams P; Walsh E; Huynh T; Chamat S; Reff M; Owyang A; Shestowsky W; Newman R

IDEC Pharmaceuticals Corporation, San Diego, California 92121, USA.

Molecular medicine (Cambridge, Mass.) (UNITED STATES) Jan 1999, 5 (1) p35-45, ISSN 1076-1551 Journal Code: 9501023

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

BACKGROUND: Respiratory syncytial virus (RSV) *infection* is a major problem in the newborn and aging populations. Fully human monoclonal antibodies with the ability to neutralize RSV could have a major impact...

... antibodies has been difficult because there exists no general way to activate B cells against an antigen of choice in vitro. MATERIALS AND METHODS: Human *spleen* cells from individuals exposed to RSV were used to repopulate SCID mice. Hu-SCID mice were boosted with RSV fusion (F)-protein and subsequently developed B cell tumors. The tumors were removed and cultured and subcloned in vitro, using a feeder layer of *CD154*-expressing T cells. Two of these tumors produced the antibodies designated RF-1 and RF-2. VL genes were isolated by standard PCR techniques, however...

... were both found to be closely related members of the VH2 family. Vk genes originated from the VK III family. RF-1 and RF-2 *recombinant* antibodies expressed in CHO cells (cRF-1 and cRF-2) were found to have affinities for RSV F-protein of 0.1 nM and 0...

...to neutralize several A and B subtypes of RSV. CONCLUSION: The technique of immortalizing human B lymphocytes, by passage in SCID mice and expression as *recombinant* antibodies in CHO cells, provides a method by which high-affinity human antibodies can be developed for immunotherapy of viral diseases.

4/3,K/2 (Item 2 from file: 155)

DIALOG(R)File 155:MEDLINE(R)
(c) format only 2004 The Dialog Corp. All rts. reserv.

14140410 PMID: 9834255

***Recombinant* *CD40L* treatment protects allogeneic murine *bone* marrow**

transplant recipients from death caused by herpes simplex virus-1 *infection*.

Beland J L; Adler H; Del-Pan N C; Kozlow W; Sung J; Fanslow W; Rimm I J

Department of Pediatric Oncology, Dana-Farber Cancer Institute and Children's Hospital, Harvard Medical School, Boston, MA, USA.

Blood (UNITED STATES) Dec 1 1998, 92 (11) p4472-8, ISSN 0006-4971 Journal Code: 7603509

Contract/Grant No.: PO1-CA39542; CA; NCI

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

***Recombinant* *CD40L* treatment protects allogeneic murine *bone* marrow transplant recipients from death caused by herpes simplex virus-1 *infection*.**

4/3,K/3 (Item 3 from file: 155)

DIALOG(R)File 155:MEDLINE(R)

12486357 PMID: 12810728

Membrane-anchored CD40 is processed by the tumor necrosis factor-alpha-converting enzyme. Implications for CD40 signaling.

Contin Cecile; Pitard Vincent; Itai Toshimitsu; Nagata Shigekazu; Moreau Jean-Francois; Dechanet-Merville Julie

CNRS UMR 5540, IFR 66, Universite Bordeaux 2, 146 rue Leo Saignat, 33076 Bordeaux, France.

Journal of biological chemistry (United States) Aug 29 2003, 278 (35) p32801-9, ISSN 0021-9258 Journal Code: 2985121R

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

The soluble form of CD40 (sCD40), which co-exists with the membrane-anchored form (mCD40), is a natural antagonist of mCD40/*CD154* interaction. However, the mechanism leading to the production of sCD40 has never been investigated. Here, we show that the engagement of mCD40 on the surface...

... traffic inhibitors but was completely blocked by a broad-spectrum synthetic metalloproteinase (MP) inhibitor (GM6001) or a membrane-anchored MP-specific inhibitor (dec-RVKR-cmk). *Recombinant* MP disintegrin tumor necrosis factor-alpha converting enzyme (TACE) cleaved the purified CD40 ectodomain/Fc chimeric protein in vitro, giving rise to an sCD40 form similar to that shed from B cell cultures. Moreover, spontaneous production of sCD40 by mCD40-transfected human embryonic *kidney* cells (constitutively expressing TACE) was enhanced by the overexpression of TACE and abrogated by co-*transfection* with a dominant-negative TACE mutant. These results provide strong evidence that sCD40 production is an active process regulated by the engagement of mCD40 and its proteolytic cleavage by TACE or a related MP disintegrin. Given the antagonistic activity of sCD40 on the CD40/*CD154* interaction, this shedding mechanism might represent an important negative feedback control of CD40 functions.

4/3,K/4 (Item 4 from file: 155)

DIALOG(R)File 155:MEDLINE(R)

12248808 PMID: 12595434

Functional CD40 expression induced following bacterial infection of mouse and human osteoblasts.

Schrum Laura W; Marriott Ian; Butler Betsy R; Thomas Elaine K; Hudson Michael C; Bost Kenneth L

Department of Biology, University of North Carolina at Charlotte, Charlotte, North Carolina 28223, USA. lwschrum@email.uncc.edu
Infection and immunity (United States) Mar 2003, 71 (3) p1209-16, ISSN 0019-9567 Journal Code: 0246127
Contract/Grant No.: AR47585; AR; NIAMS; GM58042; GM; NIGMS
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

Bacterially induced *bone* infections often result in significant local inflammatory responses which are coupled with loss of *bone*. However, the mechanisms necessary for the protective host response, or those responsible for pathogen-induced *bone* loss, are not clear. Recent evidence demonstrates that bacterially infected osteoblasts secrete chemokines and cytokines, suggesting that these cells may have an unappreciated role in... ... localized inflammation. In this study, mouse and human osteoblasts were investigated for their ability to express functional CD40 upon exposure to two important pathogens of *bone*, Staphylococcus aureus and Salmonella enterica serovar Dublin. Bacterial *infection* of cultured mouse or human osteoblasts resulted in increased CD40 mRNA and CD40 protein expression induced by either pathogen. Importantly, CD40 expression by osteoblasts was functional, as assessed by ligation of this molecule with *recombinant*, soluble *CD154* . CD40 activity was assessed by induction of interleukin-6 and granulocyte-macrophage colony-stimulating factor in osteoblasts following ligation. Cocultures of activated CD4(+) T lymphocytes and osteoblasts could interact via CD40 and *CD154*, since an antibody against CD40 could block macrophage inflammatory protein-1alpha secretion. Taken together, these studies conclusively demonstrate that infected osteoblasts can upregulate expression of functional CD40 molecules which mediate cytokine secretion. This surprising result further supports the notion that *bone*-forming osteoblasts can directly interact with *CD154*-expressing cells (i.e., T lymphocytes) and can contribute to the host response during *bone* *infection*.

4/3,K/5 (Item 5 from file: 155)

11384365 PMID: 11350957
Characterization of a novel proapoptotic caspase-2- and caspase-9-binding protein.
Bonfoco E; Li E; Kolbinger F; Cooper N R
Department of Immunology, The Scripps Research Institute, La Jolla, California 92037, USA. ebonfoco@scripps.edu
Journal of biological chemistry (United States) Aug 3 2001, 276 (31) p29242-50, ISSN 0021-9258 Journal Code: 2985121R
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

... sites. PACAP mRNA was widely expressed in most human tissues; in transfected cells, PACAP was diffusely expressed in the cytoplasm. Bindings studies with the PACAP *recombinant* protein demonstrated specific binding to casp-2 and casp-9 but not to casp-3, -4, -7, or -8 in cell extracts. Cotransfection experiments showed...

... cells. In addition, studies with truncated PACAP demonstrated a requirement for residues 39-72 of PACAP for specific binding to casp-2 and -9. Transient *transfection* of PACAP into 293T human *kidney* cells and rat-1 fibroblasts triggered apoptosis at 24 h, which was at least in part prevented by an inhibitor of casp-3-like enzymes. *Transfection* of PACAP into human B cell lines using a retroviral system also triggered apoptotic cell death. In addition, transcription of PACAP in primary human B cells was dramatically down-regulated early after cellular activation by *CD40L*

and Staphylococcus aureus and markedly up-regulated as the cells apoptose. These findings identify a novel proapoptotic caspase adaptor protein.

4/3,K/6 (Item 6 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

10945202 PMID: 11090063
Ligation of CD40 induces the expression of vascular endothelial growth factor by endothelial cells and monocytes and promotes angiogenesis in vivo.
Melter M; Reinders M E; Sho M; Pal S; Geehan C; Denton M D; Mukhopadhyay D; Briscoe D M
The Division of Nephrology, Department of Medicine, Children's Hospital, Longwood, MA 02215, USA.
Blood (UNITED STATES) Dec 1 2000, 96 (12) p3801-8, ISSN 0006-4971 Journal Code: 7603509
Contract/Grant No.: AI46756; AI; NIAID; DK53606; DK; NIDDK
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

This study addresses a mechanism by which lymphocytes may promote vascular *endothelial* growth factor (VEGF) expression and angiogenesis in immune inflammation. Resting human umbilical *endothelial* cells (HUVECs) were found to express low levels of VEGF messenger RNA (mRNA) by reverse transcription polymerase chain reaction and ribonuclease protection assay with little...

... ligand (sCD40L) resulted in a marked dose-dependent induction of VEGF mRNA (approximately 4-fold), which peaked between 1 and 5 hours post-stimulation. Transient *transfection* of HUVECs was performed with a luciferase reporter construct under the control of the human VEGF promoter. Treatment of transfected HUVECs with sCD40L was found...

...in vitro to promote growth and proliferation in a VEGF-dependent manner, and CD40-dependent HUVEC growth was comparable to that found following treatment with *recombinant* human VEGF. Furthermore, subcutaneous injection of sCD40L in severe combined immunodeficient and nude mice induced VEGF expression and marked angiogenesis in vivo. Taken together, these findings are consistent with a function for *CD40L*-CD40 interactions in VEGF-induced angiogenesis and define a mechanistic link between the immune response and angiogenesis. (Blood. 2000;96:3801-3808)

4/3,K/7 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0014781945 BIOSIS NO.: 200400148606
Rapid disease progression precludes autologous tumor vaccination in patients with relapsed ALL.
AUTHOR: Haining W Nicholas (Reprint); Cardoso Angelo A; Fleming M; Neuberg D S; De Angelo D J; Silverman L B (Reprint); Stone R M; Sallan S E (Reprint); Guinan E C (Reprint); Nadler L M
AUTHOR ADDRESS: Pediatric Oncology, Dana-Farber Cancer Institute, Boston, MA, USA**USA
JOURNAL: Blood 102 (11): p882a November 16, 2003 2003
MEDIUM: print
CONFERENCE/MEETING: 45th Annual Meeting of the American Society of Hematology San Diego, CA, USA December 06-09, 2003; 20031206
SPONSOR: American Society of Hematology
ISSN: 0006-4971
DOCUMENT TYPE: Meeting; Meeting Poster; Meeting Abstract
RECORD TYPE: Abstract

LANGUAGE: English

...ABSTRACT: therefore enrolled nine adults and children with relapsed BCP ALL in a phase I trial of CD40-ALL vaccination. Autologous ALL cells were cultured with *recombinant* human (rh) *CD40L* and rhIL4 for 4 days, and cryopreserved. Prior to vaccination, CD40-ALL cells were thawed, pulsed with keyhole limpet hemocyanin to augment T cell help...

...of either 107 cells/dose or 108 cells/dose depending on yield after culture. Vaccine generation was successful in all patients, ALL cell harvest from *bone* marrow or peripheral blood yielded a median of 2X109 ALL cells. The median post culture yield was 50%, and 3 patients had sufficient vaccine to...

...chemotherapy or stem cell transplant to stabilize disease. Despite this treatment, vaccine administration was limited by disease progression. Five patients succumbed to progressive disease or *infection* before receiving their first vaccination, and the median time to progressive disease or death was 46 days (range 11-160). Two patients withdrew from the...

4/3,K/8 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0013593024 BIOSIS NO.: 200200186535
CXCL13/BCA-1 is produced by follicular lymphoma cells: Role in the accumulation and architectural organization of malignant B cells
AUTHOR: Ghia Paolo (Reprint); Husson Herve; Cardoso Angelo A; Schultze Joachim; Munoz Olivier; Strola Giuliana (Reprint); Carideo Elizabeth G; Caligaris-Cappio Federico (Reprint); Freedman Arnold S
AUTHOR ADDRESS: Laboratory of Cancer Immunology, Institute for Cancer Research and Treatment, Candiolo, TO, Italy**Italy
JOURNAL: Blood 98 (11 Part 1): p330a-331a November 16, 2001 2001
MEDIUM: print
CONFERENCE/MEETING: 43rd Annual Meeting of the American Society of Hematology, Part 1 Orlando, Florida, USA December 07-11, 2001; 20011207
SPONSOR: American Society of Hematology
ISSN: 0006-4971
DOCUMENT TYPE: Meeting; Meeting Abstract; Meeting Poster
RECORD TYPE: Abstract
LANGUAGE: English

...ABSTRACT: architecture and cytologic features of the normal secondary lymphoid follicle. In virtually all patients, FLs are widely disseminated diseases, involving lymphoid secondary tissues and the *bone* marrow (BM). The mechanisms driving tissue localization of FL remains unclear. Chemoattractant cytokines (chemokines) play an important role in the localization of normal B lymphocytes within tissues. The chemokine CXCL13/BCA-1 is strongly expressed in the follicles of Peyer's patches, the *spleen* and lymph nodes, and is thought to be produced by follicular dendritic cells (FDCs) and follicular stromal cells. CXCL13 specifically binds to the chemokine receptor...

...of CXCL13 protein, as measured by ELISA. CXCL13 production could be significantly increased in malignant (but not in normal) B cells, after stimulation with soluble *CD40L* (sCD40L) +/- IL-4 for three days. These stimuli are known to prolong survival of both FL cells and GC-B lymphocytes in vitro. We then performed a migration assay, using *recombinant* CXCL12/SDF-1 as positive control, *recombinant* CXCL13, or the combination of both. We confirmed that all our FL samples expressed both CXCR5 (CXCL13 specific receptor) and CXCR4 (CXCL12 specific receptor). FL...

...addition to stromal cells and FDCs, and the absence of CXCL13 production by normal GCB lymphocytes suggests that this release might be associated

with neoplastic *transformation*. The fact that FL cells do express CXCR5, the CXCL13 specific receptor, and migrate in response to this chemokine, indicates the possible existence of an...
?ds

Set	Items	Description
S1	1378	(CD154 OR CD40L) (S) (SKIN OR MUSCLE OR FIBROBLAST OR ENDOTHELIAL OR NEURONAL OR BONE OR CARTILAGE OR LIVER OR KIDNEY OR SPLEEN)
S2	152	S1 (S) (TRANSFECTION OR TRANSFORMATION OR INFECTION)
S3	19	S2 (S) (RECOMBINANT)
S4	8	RD (unique items)

?s s2 not py>1999

	152	S2
	6634990	PY>1999
S5	37	S2 NOT PY>1999

?rd
...completed examining records

S6	15	RD (unique items)

?t s6/3,k/all

6/3,K/1 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

14392015 PMID: 10385635
Liver and bile duct pathology following Cryptosporidium parvum infection of immunodeficient mice.
Stephens J; Cosyns M; Jones M; Hayward A
Departments of Pathology, Pediatrics, Immunology, and the Barbara Davis Childhood Diabetes Center, University of Colorado Health Sciences Center, Denver, CO, USA.
Hepatology (Baltimore, Md.) (UNITED STATES) Jul 1999, 30 (1) p27-35, ISSN 0270-9139 Journal Code: 8302946
Contract/Grant No.: 40870; PHS; AI 41075; AI; NIAID
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

Patients with acquired immune deficiency syndrome (AIDS) and boys with mutations of the *CD154* gene (causing congenital X-linked immunodeficiency with hyper-IgM [XHIM]) are susceptible to chronic infections of the biliary tract with Cryptosporidium parvum (CP) that may lead to biliary sclerosis and ultimately to cholangiocarcinoma. To determine whether the CP *infection* and the consequent immune response contribute independently to this morbidity, we infected mice with severe combined immunodeficiency (SCID) or with disrupted genes for *CD154*, CD40, or interferon gamma (IFN-gamma) with CP. Even when CP *infection* persisted for 16 weeks, the SCID mice developed only mild triaditis, without apoptosis of biliary epithelial cells (BEC). Fifty percent of the *CD154* knockout mice developed lobular hepatitis with acute and chronic triaditis. The CD40 knockout mice developed marked triaditis, and the IFN-gamma knockouts either succumbed to...

...with periductular sclerosis, and scarring. Mice with disruptions of both the CD40 and IFN-gamma genes remained infected by CP and developed bile duct and *liver* disease, but not enteritis. Our results suggest that T-cell cytokines are required for the inflammatory and sclerosing responses to CP *infection* in immunodeficient animals. The response of immunodeficient mice to CP *infection* may model at least the initial steps toward the development of sclerosing cholangitis or bile duct cancers in XHIM patients.

6/3,K/2 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

14378221 PMID: 10374963

African swine fever virus: a B cell-mitogenic virus in vivo and in vitro.

Takamatsu H; Denyer M S; Oura C; Childerstone A; Andersen J K; Pullen L; Parkhouse R M

Institute for Animal Health, Pirbright Laboratory, Surrey, UK.

Journal of general virology (ENGLAND) Jun 1999, 80 (Pt 6) p1453-61, ISSN 0022-1317 Journal Code: 0077340

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

...of ASFV on porcine B cells have therefore been systematically examined in vivo, by using virus-infected pigs and SCID-Beige mice reconstituted with porcine *bone* marrow, and in vitro, by using porcine B-cell lines and B cells from normal and ASFV-infected pigs. Secretion of porcine Ig was stimulated by ASFV both in vivo and in *bone* marrow cultures in vitro, with the virulent Malawi isolate of ASFV being the most effective. Stimulation of Ig secretion in vitro depended on the presence...

... with supernatants from ASFV-infected macrophages. Although the virus alone did not stimulate proliferation of purified B cells in vitro, it was co-stimulatory with *CD154* (CD40 ligand). The B cells recovered from ASFV-infected porcine lymphoid tissue were of activated surface marker phenotypes and, interestingly, expressed diminished levels of the B-cell co-stimulatory surface molecule CD21. In addition, they were highly sensitive to IL-4 and *CD154*. These results may be integrated into a model of pathogenesis in which those B cells activated indirectly as a result of virulent ASFV *infection* of macrophages are not rescued from apoptosis through interaction with *CD154*, due to the drastic depletion of T cells that occurs early in *infection* . The consequently diminished specific anti-ASFV antibody response would favour survival of the virus, with the non-specific hypergammaglobulinaemia being perhaps another example of pathogen...

6/3,K/3 (Item 3 from file: 155)

14341946 PMID: 10333122

Immunological reconstitution by allogeneic bone marrow transplantation in a child with the X-linked hyper-IgM syndrome.

Kawai S; Sasahara Y; Minegishi M; Tsuchiya S; Fujie H; Ohashi Y; Kumaki S ; Konno T

Department of Paediatric Oncology, Institute of Development, Aging and Cancer, Tohoku University, Sendai, Japan.

European journal of pediatrics (GERMANY) May 1999, 158 (5) p394-7, ISSN 0340-6199 Journal Code: 7603873

Document type: Case Reports; Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

... is reported. Engraftment of HLA-identical marrow cells was obtained, although complicated by grade I acute graft-versus-host disease. Expression of the CD40 ligand (*CD40L*, *CD154*) by activated T-cells from the recipient remained at low levels until 10 months after the transplantation, but then normalized. The patient is now fully competent in immune function without any episodes of severe *infection* 24 months later. CONCLUSION: Allogeneic *bone* marrow transplantation is a reasonable therapeutic option for X-linked hyper-IgM syndrome if HLA-matched family donors are available. Whether dysregulation of *CD40L* expression causes post-transplant immunological abnormalities remains to be clarified.

6/3,K/4 (Item 4 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

14265758 PMID: 10085038
CD40 ligation prevents Trypanosoma cruzi infection through interleukin-12 upregulation.
Chaussabel D; Jacobs F; de Jonge J; de Veerman M; Carlier Y; Thielemans K ; Goldman M; Vray B
Laboratoire d'Immunologie Experimentale, Vrije Universiteit Brussel, Brussels, Belgium.
Infection and immunity (UNITED STATES) Apr 1999, 67 (4) p1929-34, ISSN 0019-9567 Journal Code: 0246127
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

Because of the critical role of the CD40-CD40 ligand (*CD40L*) pathway in the induction and effector phases of immune responses, we investigated the effects of CD40 ligation on the control of Trypanosoma cruzi *infection*. First, we observed that supernatants of murine *spleen* cells stimulated by *CD40L*-transfected 3T3 fibroblasts (3T3-*CD40L* transfectants) prevent the *infection* of mouse peritoneal macrophages (MPM) by T. cruzi. This phenomenon depends on de novo production of nitric oxide (NO) as it is prevented by the...

...MAb). We found that an activating anti-CD40 MAb also directly stimulates IFN-gamma-activated MPM to produce NO and thereby to control T. cruzi *infection*. To determine the in vivo relevance of these in vitro findings, mice were injected with 3T3-*CD40L* transfectants or 3T3 control fibroblasts at the time of T. cruzi inoculation. We observed that in vivo CD40 ligation dramatically reduced both parasitemia and the mortality rate of T. cruzi-infected mice. A reduced parasitemia was still observed when the injection of 3T3-*CD40L* transfectants was delayed 8 days postinfection. It was abolished by injection of anti-IL-12 MAb. Taken together, these data establish that CD40 ligation facilitates the control of T. cruzi *infection* through a cascade involving IL-12, IFN-gamma, and NO.

6/3,K/5 (Item 5 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

14253416 PMID: 10072446
Two neutralizing human anti-RSV antibodies: cloning, expression, and characterization.
Heard C; Brams P; Walsh E; Huynh T; Chamat S; Reff M; Owyang A; Shestowsky W; Newman R
IDEC Pharmaceuticals Corporation, San Diego, California 92121, USA.
Molecular medicine (Cambridge, Mass.) (UNITED STATES) Jan 1999, 5 (1) p35-45, ISSN 1076-1551 Journal Code: 9501023
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

BACKGROUND: Respiratory syncytial virus (RSV) *infection* is a major problem in the newborn and aging populations. Fully human monoclonal antibodies with the ability to neutralize RSV could have a major impact...

... antibodies has been difficult because there exists no general way to activate B cells against an antigen of choice in vitro. MATERIALS AND METHODS: Human *spleen* cells from individuals exposed to RSV were used to repopulate SCID mice. Hu-SCID mice were boosted with RSV fusion (F)-protein

and subsequently developed B cell tumors. The tumors were removed and cultured and subcloned in vitro, using a feeder layer of *CD154*-expressing T cells. Two of these tumors produced the antibodies designated RF-1 and RF-2. VL genes were isolated by standard PCR techniques, however...

6/3,K/6 (Item 6 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

14158939 PMID: 9857288
Leukocyte transfusion-associated granulocyte responses in a patient with X-linked hyper-IgM syndrome.
Atkinson T P; Smith C A; Hsu Y M; Garber E; Su L; Howard T H; Prchal J T; Everson M P; Cooper M D
Department of Pediatrics, University of Alabama at Birmingham 35294, USA.
Journal of clinical immunology (UNITED STATES) Nov 1998, 18 (6) p430-9, ISSN 0271-9142 Journal Code: 8102137
Document type: Case Reports; Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

X-linked hyper-IgM syndrome (XHIM) is a severe congenital immunodeficiency caused by mutations in *CD154* (CD40 ligand, gp39), the T cell ligand for CD40 on B cells. Chronic or cyclic neutropenia is a frequent complicating feature that heightens susceptibility to...

... neutropenia. Eight of ten leukocyte transfusions with cells from a maternal aunt, performed because of mucosal infections, resulted in similar episodes of endogenous granulocyte production. *Transfection* studies with the mutant *CD154* protein indicate that the protein is expressed at the cell surface and forms an aberrant trimer that does not interact with CD40. The data suggest that allogeneic cells from the patient's aunt, probably activated T cells bearing functional *CD154* , may interact with CD40+ recipient cells to produce maturation of myeloid precursors in the *bone* marrow.

6/3,K/7 (Item 7 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

14140410 PMID: 9834255
Recombinant *CD40L* treatment protects allogeneic murine *bone* marrow transplant recipients from death caused by herpes simplex virus-1 *infection*.
Beland J L; Adler H; Del-Pan N C; Kozlow W; Sung J; Fanslow W; Rimm I J
Department of Pediatric Oncology, Dana-Farber Cancer Institute and Children's Hospital, Harvard Medical School, Boston, MA, USA.
Blood (UNITED STATES) Dec 1 1998, 92 (11) p4472-8, ISSN 0006-4971 Journal Code: 7603509
Contract/Grant No.: PO1-CA39542; CA; NCI
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

Recombinant *CD40L* treatment protects allogeneic murine *bone* marrow transplant recipients from death caused by herpes simplex virus-1 *infection*.
Posttransplant *infection* associated with host immune deficiency is the major cause of nonrelapse mortality of human *bone* marrow transplant recipients. In a new murine model of posttransplant *infection*, allogeneic *bone* marrow transplant recipients were infected with herpes simplex virus-1 (HSV-1) via intraperitoneal inoculation 12 weeks after transplantation. Allogeneic transplant recipients with graft-versus...

...both T-cell and B-cell defects contributed to the impaired production of antibody. Because passive transfer of hyperimmune serum protected mice from HSV-1 *infection*, we hypothesized that CD40 ligand (*CD40L*), which induces B-cell maturation, would protect mice from HSV-1 *infection*. *CD40L* -treated GVHD mice showed elevated IgG2a levels and increased survival compared with vehicle-treated transplant recipients.

6/3,K/8 (Item 8 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

13825147 PMID: 9525309
Transient immunosuppression allows transgene expression following readministration of adeno-associated viral vectors.
Manning W C; Zhou S; Bland M P; Escobedo J A; Dwarki V
Chiron Corporation, Emeryville, CA 94608, USA.
Human gene therapy (UNITED STATES) Mar 1 1998, 9 (4) p477-85, ISSN 1043-0342 Journal Code: 9008950
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

... vector for gene therapy are its ability to infect both dividing and nondividing cells and the longevity of expression in tissues such as brain, skeletal *muscle*, and *liver* . However, like other viral vectors, readministration of vector is limited because of the host's immune response to viral components of the vector. Using class I, class II, and CD40 ligand (*CD40L*)-deficient mice, we demonstrate that neutralizing antibodies to the viral capsid proteins prevent transgene expression following readministration of rAAV vectors. Transient immunosuppression of mice by treatment with antibody to CD4 at the time of primary *infection* allowed transgene expression after readministration of rAAV vectors to animals. Transient immunosuppression with antibody to *CD40L* had only a modest effect on the efficacy of readministration. The ability to readminister virus was inversely correlated with both AAV capsid enzyme-linked immunosorbent...

6/3,K/9 (Item 9 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

13802564 PMID: 9499800
Evidence for a continued requirement for CD40/CD40 ligand (CD154) interactions in the progression of LP-BM5 retrovirus-induced murine AIDS.
Green K A; Noelle R J; Green W R
Department of Microbiology, Dartmouth Medical School, Lebanon, New Hampshire 03756, USA.
Virology (UNITED STATES) Feb 15 1998, 241 (2) p260-8, ISSN 0042-6822 Journal Code: 0110674
Contract/Grant No.: CA-23108; CA; NCI; CA50157; CA; NCI
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

... of MAIDS is dependent on the presence of both CD4+ T and B cells, we have previously shown that anti-gp39/CD40 ligand mAb (anti-*CD40L* mAb) treatment of LP-BM5-infected mice is effective in inhibiting the induction of MAIDS when a short course of anti-*CD40L* mAb treatment was started on the same day as LP-BM5 administration. The success of anti-*CD40L* mAb therapy, as indicated by a much reduced degree of splenomegaly, hypergammaglobulinemia, and mitogen and allogeneic CTL unresponsiveness, demonstrated that *CD40L* /CD40 interactions were critical to the

establishment of MAIDS. Here we extend these findings through the use of delayed anti-*CD40L* mAb treatment of mice, beginning 3-4 weeks after LP-BM5 *infection*, by showing that interruption of *CD40L* /CD40 interactions also interferes with the progression of MAIDS. About 60% of LP-BM5-preinfected mice were affected by delayed anti-*CD40L* mAb treatment, with substantially reduced *spleen* weights and serum hypergammaglobulinemia and normal or greatly restored proliferative responses to Con A stimulation and CTL responses to allogeneic stimulation. The other LP-BM5-infected mice that did not respond to anti-*CD40L* therapy were found to have made antibodies to the anti-*CD40L* mAb. Thus, in a majority of mice anti-*CD40L* mAb therapy was very effective in interfering with MAIDS pathogenesis well after the establishment of the virus *infection* and MAIDS symptomatology, indicating that *CD40L* /CD40 interactions are crucial to the maintenance and progression of the disease, as well as its initiation.

6/3,K/10 (Item 10 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

13800349 PMID: 9498737
CD40 ligand is not essential for the development of cell-mediated immunity and resistance to Mycobacterium tuberculosis.
Campos-Neto A; Ovendale P; Bement T; Koppi T A; Fanslow W C; Rossi M A; Alderson M R
Infectious Disease Research Institute, Seattle, WA 98104, USA. acampos@corixa.com
Journal of immunology (Baltimore, Md. - 1950) (UNITED STATES) Mar 1 1998, 160 (5) p2037-41, ISSN 0022-1767 Journal Code: 2985117R
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

It has been proposed that the induction of cellular immunity and resistance to intracellular pathogens is dependent upon CD40 ligand (*CD40L*). In the present study we show that this proposal is not ubiquitously supported. Mice genetically deficient in *CD40L* (CD40LKO) were resistant to i.v. *infection* with Mycobacterium tuberculosis when assessed by survival and bacteriologic burden in the *spleen*, *liver*, and lungs. Infected CD40LKO mice developed granulomas that lacked epithelioid cells and were less numerous and markedly smaller than those observed in control mice. Upon...

... protein derivative of M. tuberculosis, CD4+ T cells from infected CD40LKO mice proliferated and produced high levels of IFN-gamma but not IL-4. Finally, *spleen* cells from CD40LKO mice stimulated with M. tuberculosis produced IL-12, TNF, and nitric oxide levels comparable to those produced by control cells. In contrast to original proposals, these data clearly show that protective Th1 immunity can be achieved against intracellular pathogens (e.g., Mycobacterium) independently of *CD40L*.

6/3,K/11 (Item 11 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

13757499 PMID: 9453615
Requirement of CD40-CD40 ligand interaction for elimination of Cryptosporidium parvum from mice.
Cosyns M; Tsirkin S; Jones M; Flavell R; Kikutani H; Hayward A R
Department of Pediatrics and Immunology, University of Colorado School of Medicine, Denver 80262, USA. Mary.Cosyns@uchsc.edu
Infection and immunity (UNITED STATES) Feb 1998, 66 (2) p603-7, ISSN 0019-9567 Journal Code: 0246127
Document type: Journal Article

Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

Mice with disrupted genes for CD40 and CD40 ligand (*CD40L*) are unable to clear *infection* with Cryptosporidium parvum and develop cholangitis. Parasites are present in the gut, gall bladder, and biliary tree, and biliary epithelial cells express CD40 on the cell surface. SCID mice infected with C. parvum for >1 month can clear the *infection* after reconstitution with *spleen* cells from CD40, but not *CD40L*, knockout mice. In an in vitro model, C. parvum-infected HepG2 cells were triggered to apoptosis when incubated with a *CD40L* -CD8 fusion protein. The requirement for CD40-*CD40L* interactions for immunity to C. parvum indicated by our results may entail the triggering of apoptosis in infected cells, in addition to the known role of *CD40L*-CD40 interactions in stimulating cytokine production and promoting T-cell responses.

6/3,K/12 (Item 12 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

13668206 PMID: 9359699
HIV-1 induction of CD40 on endothelial cells promotes the outgrowth of AIDS-associated B-cell lymphomas.
Moses A V; Williams S E; Strussenberg J G; Heneveld M L; Ruhl R A; Bakke A C; Bagby G C; Nelson J A
Department of Molecular Microbiology and Immunology, Oregon Health Sciences University, Portland 97201-3011, USA.
Nature medicine (UNITED STATES) Nov 1997, 3 (11) p1242-9, ISSN 1078-8956 Journal Code: 9502015
Contract/Grant No.: DK 49887; DK; NIDDK; MH 51519; MH; NIMH
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

Human immunodeficiency virus (HIV)-1 *infection* is associated with the development of aggressive extranodal B-cell non-Hodgkin's lymphomas. Using microvascular *endothelial* cell (MVEC)-enriched *bone* marrow stromal cultures, HIV *infection* of stromal MVECs from lymphoma patients induced the outgrowth of malignant B cells. MVECs were the only HIV-infected cells in the stroma, and purified brain MVECs also induced a phenotype supportive of neoplastic B-cell attachment and proliferation. HIV *infection* of MVECs stimulated surface expression of CD40 and allowed preferential induction of the vascular cell adhesion molecule VCAM-1 after CD40 triggering. B-lymphoma cells expressed the CD40 ligand (*CD40L*), and blocking of CD40-*CD40L* interactions between HIV-infected MVECs and B-lymphoma cells inhibited B-cell attachment and proliferation. These observations suggest that HIV promotes B-lymphoma cell growth...

6/3,K/13 (Item 13 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

12772481 PMID: 7561048
CD40 ligand is required for resolution of Pneumocystis carinii pneumonia in mice.
Wiley J A; Harmsen A G
Trudeau Institute, Inc., Saranac Lake, NY 12983, USA.
Journal of immunology (Baltimore, Md. - 1950) (UNITED STATES) Oct 1 1995, 155 (7) p3525-9, ISSN 0022-1767 Journal Code: 2985117R
Contract/Grant No.: AI-28354; AI; NIAID
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM

Record type: Completed

The role of the CD40-CD40 ligand (*CD40L*) interaction in resolution of Pneumocystis carinii (PC) pneumonia (PCP) was assessed in a PC-infected severe combined immunodeficiency (SCID) mouse reconstitution model using an anti-*CD40L* mAb to block *CD40L* . SCID mice infected with PC were reconstituted with unfractionated *spleen* cells from immunocompetent donors and given either anti-*CD40L* mAb or an irrelevant control mAb. Mice given the control mAb resolved the PC *infection*, whereas those given the anti-*CD40L* mAb did not. That anti-*CD40L* mAb also inhibited PC-specific IgG production is consistent with the possibility that cognate CD4+ T cell-B cell interactions are important in PCP resolution...

... infected SCID mice were reconstituted with purified CD4+ T cells only. Again, the control mAb-treated group resolved the PCP, whereas mice treated with anti-*CD40L* mAb did not. In the second experiment, inhibition of resolution of PCP in the anti-*CD40L* mAb group was not the result of blocking CD4+ T cell-dependent activation of PC-specific B cells. The results are consistent with the possibility that resistance to PCP may involve interaction between B cells and CD4+ T cells via the CD40-*CD40L* pathway. However, results additionally indicate that inhibition of CD40-*CD40L* interaction ablates resistance to PCP by inhibiting the interaction of T cells with some cell other than B cells.

6/3,K/14 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE

07207322 EMBASE No: 1998085680
Cutting edge: CD40 ligand is not essential for the development of cell-mediated immunity and resistance to Mycobacterium tuberculosis
Campos-Neto A.; Ovendale P.; Bement T.; Koppi T.A.; Fanslow W.C.; Rossi M.A.; Alderson M.R.
Dr. A. Campos-Neto, Infectious Disease Res. Institute, 1124 Columbia St., Seattle, WA 98104 United States
AUTHOR EMAIL: acampos@corixa.com
Journal of Immunology (J. IMMUNOL.) (United States) 01 MAR 1998, 160/5 (2037-2041)
CODEN: JOIMA ISSN: 0022-1767
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 29

It has been proposed that the induction of cellular immunity and resistance so intracellular pathogens is dependent upon CD40 ligand (*CD40L*). In the present study we show that this proposal is not ubiquitously supported. Mice genetically deficient in *CD40L* (CD40LKO) were resistant to i.v. *infection* with Mycobacterium tuberculosis when assessed by survival and bacteriologic burden in the *spleen*, *liver*, and lungs. Infected CD40LKO mice developed granulomas that lacked epithelioid cells and were less numerous and markedly smaller than those observed to control mice. Upon...

...protein derivative of M. tuberculosis, CD4sup + T cells from infected CD40LKO mice proliferated and produced high levels of IFN-gamma but not IL-4. Finally, *spleen* cells from CD40LKO mice stimulated with M. tuberculosis produced IL-12, TNF, and nitric oxide levels comparable to those produced by control cells. In contrast to original proposals, these data clearly show that protective Th1 immunity can be achieved against intracellular pathogens (e.g., Mycobacterium) independently of *CD40L*.

6/3,K/15 (Item 2 from file: 73)
DIALOG(R)File 73:EMBASE

07137304 EMBASE No: 1998023162

CD40 ligand expression in mycosis fungoides is restricted to the patch/plaque (epidermotropic) stages

Mori M.; Manuelli C.; Pimpinelli N.; Mavilia C.; Bianchi B.; Santucci M.; Giannotti B.; Cappugi P.

M. Mori, Inst. of Dermatology and Venereology, Univ. of Florence Medical School, via degli Alfani 37, 50121 Florence Italy

European Journal of Dermatology (EUR. J. DERMATOL.) (France) 1997, 7/8 (556-560)

CODEN: EJDEE ISSN: 1167-1122

DOCUMENT TYPE: Journal; Article

LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

NUMBER OF REFERENCES: 34

CD40 ligand (*CD40L*) is a member of the tumor necrosis factor ligand superfamily. It may play an intricate role in the immune response by functional interaction with CD40 antigen expressed on the surface of B-cells, T-cells, monocytes, macrophages, antigen-presenting cells, *endothelial* cells and some epithelial cells. A possible role of *CD40L* in the pathogenesis of non-Hodgkin's lymphoma (NHL) has been recently hypothesized; in fact, *CD40L* antigen can be constitutively expressed by the neoplastic CD4sup + T-cell of nodal NHL, thus possibly having a physiological role in these neoplasms. We studied the immunophenotypic and genotypic expression of *CD40L* antigen in different phases of mycosis fungoides - the prototype of cutaneous T-cell lymphoma - in order to investigate the possible significance and role of this...

...junctional and basal epidermal) were very similar to those of CD25sup + and Ki67sup + (proliferating) cells. In 5/5 specimens from tumor stage mycosis fungoides, no *CD40L* immunostaining was found. All patch/plaque stage mycosis fungoides (6/6 specimens) contained the mRNA transcript for *CD40L*. It was never detected in tumor stage mycosis fungoides (4/4 specimens). These findings suggest that in early mycosis fungoides, CD40Lsup + T-cells home into the *skin* by interaction with CD40sup + *endothelial* cells and into the epidermis by interaction with CD40sup + basal epidermal cells. The interaction between CD40Lsup + T-cells and CD40sup + Langerhans cells and the CD40/*CD40L* autocrine stimulus possibly triggers activation, growth and neoplastic enhancement of T-cells; up to the blastic *transformation* occurring at the tumor stage, when neoplastic T-cells loose their antigenic and functional features of mature T-cells and proliferate without any significant control.

?ds

Set	Items	Description
S1	1378	(CD154 OR CD40L) (S) (SKIN OR MUSCLE OR FIBROBLAST OR ENDOTHELIAL OR NEURONAL OR BONE OR CARTILAGE OR LIVER OR KIDNEY OR SPLEEN)
S2	152	S1 (S) (TRANSFECTION OR TRANSFORMATION OR INFECTION)
S3	19	S2 (S) (RECOMBINANT)
S4	8	RD (unique items)
S5	37	S2 NOT PY>1999
S6	15	RD (unique items)

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        136274  TRANSFORMING
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9/3,K/1 (Item 1 from file: 155)

DIALOG(R)File 155:MEDLINE(R)

13770937 PMID: 9461447

Isolation of differentially expressed genes upon immunoglobulin class switching by a subtractive hybridization method using uracil DNA glycosylase.

Sugai M; Kondo S; Shimizu A; Honjo T

Department of Medical Chemistry, Faculty of Medicine and Center for Molecular Biology and Genetics, Kyoto University Sakyo-ku, Kyoto 606, Japan.

Nucleic acids research (ENGLAND) Feb 15 1998, 26 (4) p911-8, ISSN 0305-1048 Journal Code: 0411011

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

...system using a cell line, CH12F3-2. To clarify the molecular mechanism of class switching, we intended to isolate genes induced with interleukin (IL)-4, *transforming* growth factor (TGF)-beta and *CD40L* using the in vitro class switching system. For that purpose, an improved method for making subtracted cDNA libraries, using uracil DNA glycosylase, has been developed...
... lost. This new subtraction method was applied to the CH12F3-2 switching system to isolate genes induced by stimulations with IL-4, TGF-beta and *CD40L*, and cDNAs encoding deiodinase 1 and SS32, an alternatively spliced form of the *muscle* LIM protein, were obtained. Their expression patterns in response to various combinations of stimuli and the time courses of the induction supported the usefulness of...

9/3,K/2 (Item 2 from file: 155)

DIALOG(R)File 155:MEDLINE(R)

13727127 PMID: 9422424

Blockade of the CD40-CD40 ligand pathway potentiates the capacity of donor-derived dendritic cell progenitors to induce long-term cardiac allograft survival.

Lu L; Li W; Fu F; Chambers F G; Qian S; Fung J J; Thomson A W

Thomas E. Starzl Transplantation Institute and Department of Surgery, University of Pittsburgh, Pennsylvania 15213, USA.

Transplantation (UNITED STATES) Dec 27 1997, 64 (12) p1808-15, ISSN 0041-1337 Journal Code: 0132144

Contract/Grant No.: R01 AI41011; AI; NIAID; R01 DK 49745; DK; NIDDK

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

... up-regulation of these molecules on the DCs after interaction with host T cells. Ligation of CD40 on antigen-presenting cells by its cognate ligand *CD40L* is thought to induce expression of CD80 (B7-1) and CD86 (B7-2). We examined the influence of anti-*CD40L* monoclonal antibody (mAb) on the capacity of donor-derived DC progenitors to induce long-term allograft survival. METHODS: High purity DC progenitors were grown from B10 (H2b) mouse *bone* marrow in granulocyte-macrophage colony-stimulating factor and *transforming* growth factor beta1 (TGFbeta1). Mature DC were propagated in granulocyte-macrophage colony-stimulating factor and interleukin-4. Their phenotype was characterized by flow cytometric analysis...

... days vs. 12 days in controls, and 5 days in interleukin-4 DC-treated animals). Their allostimulatory activity was further diminished by addition of anti-*CD40L* mAb at the start of the mixed leukocyte cultures. Anti-*CD40L* mAb alone (250 microg/mouse, i.p.; day -7) did not prolong cardiac

graft survival (MST 12 days). In contrast, TGFbeta-cultured DCs + anti-*CD40L* mAb extended graft survival to a MST of 77 days, and inhibited substantially the anti-donor cytotoxic T lymphocyte activity of graft-infiltrating cells and host *spleen* cells assessed 8 days after transplant. CONCLUSIONS: The CD40-*CD40L* pathway appears important in regulation of allogeneic DC-T-cell functional interaction in vivo; its blockade increases markedly the potential of costimulatory molecule-deficient DCs...

9/3,K/3 (Item 3 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

12950154 PMID: 8634441
Transforming growth factor-beta1: differential effects on multiple myeloma versus normal B cells.
Urashima M; Ogata A; Chauhan D; Hatziyanni M; Vidriales M B; Dedera D A; Schlossman R L; Anderson K C
Division of Hematologic Malignancies, Dana-Farber Cancer Institute, Boston, MA, USA.
Blood (UNITED STATES) Mar 1 1996, 87 (5) p1928-38, ISSN 0006-4971 Journal Code: 7603509
Contract/Grant No.: CA50947; CA; NCI
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

Interleukin-6 (IL-6), a product of *bone* marrow stromal cells (BMSCs), is a growth factor for multiple myeloma (MM) cells. *Transforming* growth factor-beta1 (TGF-beta1) is also produced by BMSCs and can regulate IL-6 secretion by several tissues, including BMSCs. The present study was...

... significantly more TGF-beta1 (8.2 +/- 2.0 ng/mL) than peripheral blood mononuclear cells (P < .001), splenic B cells (P < .001), and CD40 ligand (*CD40L*) pretreated B cells (P < .05). TGF-beta1 secretion by MM-BMMCs (3.8 +/- 0.9 ng/mL) was significantly greater than by N-BMMCs (1...

... constitutively phosphorylated in MM cells; TGF-beta1 either did not alter or increased pRB phosphorylation. pRB are dephosphorylated in splenic B cells and phosphorylated in *CD40L* triggered B cells in contrast to its effects on MM cells, TGF-beta1 decreased phosphorylation of pRB in *CD40L* treated B cells. These results suggest that TGF-beta1 is produced in MM by both tumor cells and BMSCs, with related tumore cell growth. Moreover...
?ds

Set	Items	Description
S1	1378	(CD154 OR CD40L) (S) (SKIN OR MUSCLE OR FIBROBLAST OR ENDOTHELIAL OR NEURONAL OR BONE OR CARTILAGE OR LIVER OR KIDNEY OR SPLEEN)
S2	152	S1 (S) (TRANSFECTION OR TRANSFORMATION OR INFECTION)
S3	19	S2 (S) (RECOMBINANT)
S4	8	RD (unique items)
S5	37	S2 NOT PY>1999
S6	15	RD (unique items)
S7	23	S1 (S) (TRANSFECTING OR TRANSFORMING)
S8	9	S7 NOT PY>1999
S9	3	RD (unique items)

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$3.50 2 Type(s) in Format 3

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      $2.49 TELNET
     $32.23 Estimated cost this search
     $32.55 Estimated total session cost    2.723 DialUnits


### Status: Signed Off. (10 minutes)
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